

Current sources of initial QRS forces in left ventricular hypertrophy, WPW syndrome, and left bundle branch block – origin of septal vector

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1 Introduction

Since the original investigations of Lewis and Rothschild (1915) [1], septal activation has been thought to occur first on the left septal surface and thereafter to spread from the left side to the right side of the intraventricular septum. Thus, the initial QRS vector in electrocardiogram is considered to be due to activation of intraventricular septum and called as septal vector. At this time (within 20 msec from the onset of the QRS wave), posterobasal area is not activated. However, normal septal vector disappears in cases of inferior infarction as well as anteroseptal infarction. Then arises the question whether the initial QRS vector is really composed of intraventricular septum alone. In left ventricular hypertrophy, the initial QRS vector becomes smaller or even absent. Although the mechanism of disappearance of septal vector in left ventricular hypertrophy is now considered to be due to septal fibrosis, the precise mechanism remains unclear [2]. Therefore, we studied the origin of initial QRS force by magnetocardiogram (MCG). The MCG three-dimensional (3-D) source localization using superconducting quantum interference device (SQUID) gradiometer has an excellent spatial and temporal resolution. To clarify the mechanism of disappearance of septal vector (QS pattern in lead V₁) in LVH, the MCG of normal subjects and the patients with left ventricular hypertrophy were recorded in an rf-shielded room using biomagnetic measuring system. We also studied the initial QRS force in patients with left bundle branch block (LBBB) and type B WPW syndrome, in which the septal vector is absent. The results of the study provide a new interpretation of ECG wave forms.

2 Methods

2.1 Subjects

We studied 20 normal subjects (N group), 33 patients with left ventricular overload (LVH group), 10 patients with complete LBBB (LBBB group) and 12 patients with type B WPW syndrome (WPW group), in which initial QRS vector is also frequently deviated. The LVH group was subdivided

into two subgroups according to the presence or absence of an initial r waves in lead V₁, so-called septal vector, i.e. those with rS pattern (LVH-rS group) and those without r wave (LVH-QS group). In the LVH-QS group, the r wave in lead V₁ and/or q wave in lead V₆ was absent. The LVH-rS group shows the r wave in lead V₁ and q wave in lead V₆. The echocardiogram and magnetic resonance imaging (MRI) were recorded in all subjects to localize anatomically current sources.

2.2 Construction of isofield map and source localization

The MCG was recorded over the anterior chest wall with a second-derivative superconducting quantum interference device (SQUID) gradiometer (single channel rf SQUID; Model BMP, BTI, San Diego, or 7-ch dc-SQUID, Liquid Gas Corp. Osaka) in an rf-shielded room at Tokushima University Hospital. The patients were placed in the supine position on a nonmagnetic bed. All metal objects, including watches, metallic contents of trousers, such as keys and hairpins, were removed. The magnetic field perpendicular to the anterior chest wall was measured. The MCG recording was taken by scanning each recording point (25 points or 63 points) with a bandpass filter of 0.02 - 300Hz. The position of detector was confirmed by laser point indicator. The reference points used were the xyphoid process and the top of the sternum. The MCG measurements were based on a three-dimensional (3-D) coordinate system consisting of a median line between the xyphoid process and the top of the sternum. The origin was defined as the xyphoid process. The Y axis was defined as the line passing up from the origin and parallel to the sagittal plane (inferior to superior); the X as the line passing out of the left side from the origin (right to left), and the Z axis as the line passing from the origin and perpendicular from the plane of the detection coil (anterior to posterior).

Isofield map at every 2 msec was constructed by using the same methods as construction of isopotential map [3]. In order to localize the current

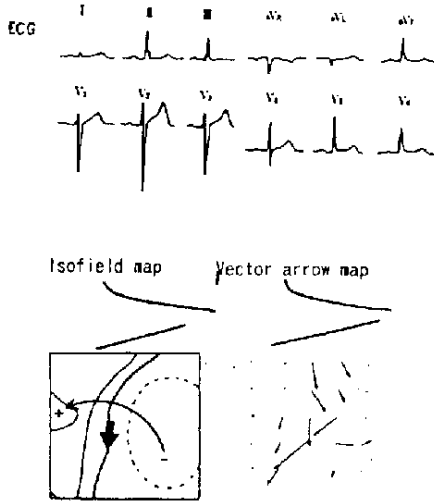


Figure 1: Isofield map and vector arrow map at 20msec in a normal subject. Initial QRS vector directed to the right and inferiorly.

dipole, a single dipole model [4,5] was used and the 3-D location of the equivalent current dipole was computed every 2ms from the onset of the QRS complex by the least square method. The 3-dimensional dipole locations were superimposed onto the MRI of the individual subjects providing anatomical localization in the ventricle.

2.3 Vector arrow map

We also constructed vector arrow maps to study the multiple instantaneous vector according to the methods by Hosaka and Cohen [6]. A vector arrow (A) was defined as follows:

$$A = (\partial B_n / \partial y) \mathbf{x} - (\partial B_n / \partial x) \mathbf{y}$$

where x and y are axes of the body, x in the direction from right to left, and y from head to foot, and $\mathbf{x}$ and $\mathbf{y}$ are vectors of unit length along the x and y axes. Vector arrow was obtained at each point. Vector arrows on the map are supposed to indicate the underlying current pattern parallel to the frontal plane.

3 Results

3.1 Initial QRS wave in normal subjects

Fig. 1 shows isofield map and vector arrow map in a normal subject. The deduced dipole of normal subjects was located on the ventricular septum in 9 of 10 subjects who recorded MRI. Mean values of deduced dipole of the initial 20 msec vector were 48 ± 18 mm left ward, 58 ± 24 mm superior to the xyphoid process and 41 ± 13 mm deep from the surface of the gradiometer. The deduced dipole at early QRS moved from mid-septum to the apical septum in most of the cases.

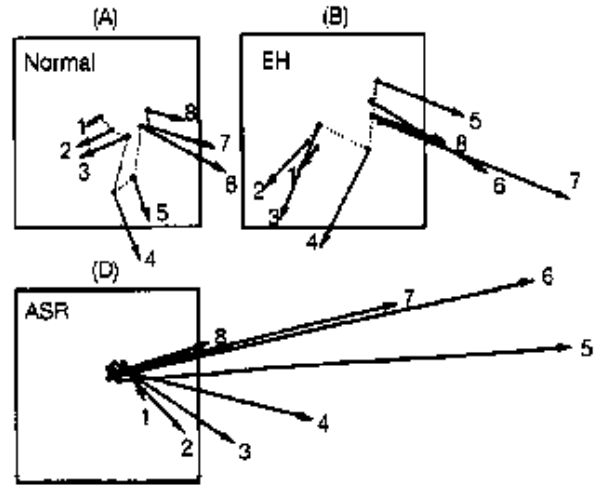


Figure 2; Single moving dipole in a normal subject (A), a patient with essential hypertension (EH) and a patient with aortic valve disease (ASR). Note that 20 msec dipole (2) directed to the left and displaced to the left in ASR.

Table 1: Location of the single dipole at 20 msec in normal subjects and patients with left ventricular hypertrophy.

Group	n	Distance from coil
Normal	32	56 ± 2 mm
LVH-rS	25	62 ± 4 mm **
LVH-QS	13	70 ± 5 mm**
WPW	12	54 ± 9 mm
LBBS	10	53 ± 5 mm

*, **; $p < 0.05$, $p < 0.01$ vs Normal

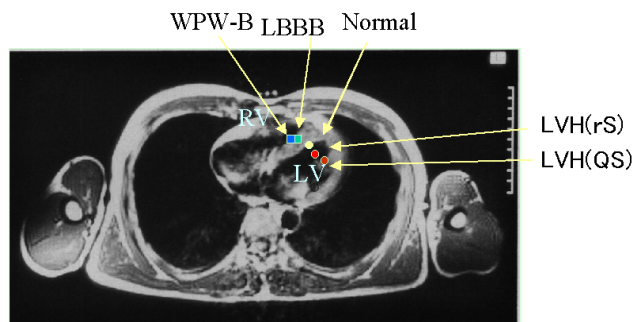


Figure 3: *Location of deduced dipole in each group on MRI.*

3.2 Initial QRS wave in patients

Fig. 2 shows the movement of single dipoles during ventricular depolarization in normal subject and patients with LVH. In patients with LVH (ARS) initial QRS dipole was displaced to the left and its direction was also displaced to the left. At 50msec further increase in vectors directing leftward was observed. Table 1 shows that the deduced current source in LVH group was displaced posteriorly and to the left. The deviation was significantly greater in the LVH-QS group than that of LVH-rS group. However, those of LBBB and WPW groups were not displaced and located similarly with that of normal subjects. Fig. 3 shows the MRI in which the dipole deduced by isofield mapping in each group was superimposed.

3.3 Analysis by vector arrow map

Fig. 4 shows the ECG, isofield map and vector arrow map of a patient with left ventricular overload with small r wave in lead V_1 but not q waves in left precordial leads (V_5 and V_6). At 20 msec Vector arrow map shows that vector arrows locating in the right shows normal vector directing rightward but those locating in the left directing to the left with increased amplitude. The dipole deduced by isofield map was directed to the left probably as a result of the summation of vector arrows of both directions. Compared to this patient the vector arrow map in a patient with LBBB (Fig.5) showed dipoles directing to the left was not increased in amplitude at the left part. In late phase of depolarization of LBBB, the increase of vector arrows in amplitude were not observed.

3 Discussion

In the present study, we determined the location of the initial QRS force in various pathological conditions in which the septal vector is absent. The present study shows that the initial QRS force deduced by the MCG was displaced posteriorly in left ventricular hypertrophy and was increased in magnitude, which were different from that of LBBB or type B WPW syndrome. These results suggest that absence of septal vector might be due to the increased electromotive forces of the hypertrophied left ventricle rather than conduction disturbance. The results of the present studies explain the mechanism of abnormal initial QRS forces in ECG.

The initial QRS force has been considered to originate from the activation of the left-sided septum which moves to the rightward. It is considered that posterobasal area of the left ventricle is activated later than septum and that this part does not contribute to generation of normal septal vector. However, we have reported that in inferior myocardial infarction initial QRS vector was

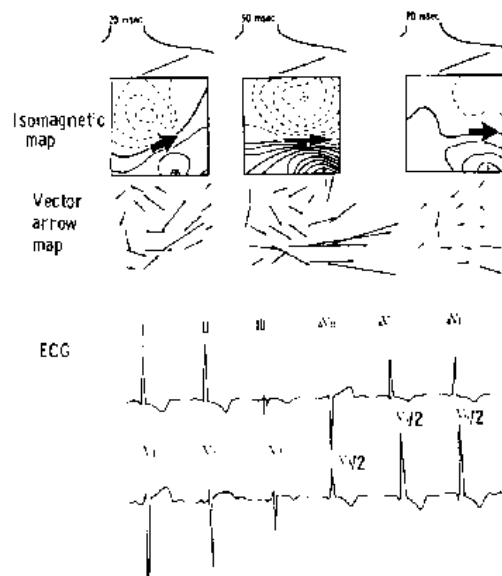


Figure 4: *Isofield map and vector arrow map of a patient with left ventricular overload with small r wave in lead V_1 . In the vector arrow map at 20 msec, dipoles located on the left are increased in amplitude and directed to the left.*

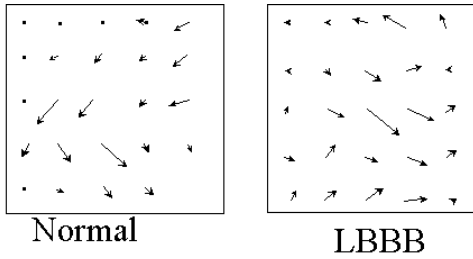


Figure 5: Vector arrow map in a normal and a patient with LBBB. Note that there is no increase in vector arrow in the left part of the map in comparison with the map in figure 4.

displaced, suggesting that posterobasal area (infarct area) also contributes initial QRS force. These findings strongly support the idea that the initial QRS vector is not originated from ventricular septum alone, and that many areas of ventricle contribute the formation of initial QRS vector.

In left ventricular hypertrophy, the initial QRS vector is frequently deviated, which was explained by septal fibrosis due to hypertrophy. The vector arrow map studies also suggested that the rightward vector directed to the left suggesting normal rightward electromotive force, although decreased in amplitude. In addition to the normal septal vector, there were increased leftward directing electromotive force in the left ventricle. Thus, the resultant QRS force as a summation of many portions of the both ventricles is located leftward and directed to the left, because the balance of the electromotive force between the left and right ventricles was altered. (Fig. 6)

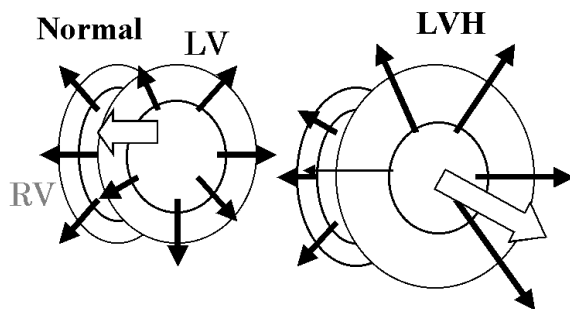


Figure 6: The mechanism of QS pattern in lead V_1 in LVH (hypothesis from this study). The initial QRS force is a summation vector of various portion of the ventricles. In LVH, hypertrophied left ventricle produces larger electromotive force directed to the left and posteriorly, resulting in displacement of the initial vector to the left and posteriorly.

The initial QRS vector has been considered to be due to the intraventricular activation. However, the present study shows that other parts of the ventricle also contribute to generate initial QRS vectors. Thus

the initial QRS vector is a hypothetical resultant or mean vector representing the average direction and magnitude of all electric forces produced at this time. In agreement with our studies, three endocardial areas are synchronously depolarized from 0-5 msec after the onset of ventricular activation in the studies of human heart [7]. These studies also support the results of the present studies that the initial QRS vector is summation of electromotive forces of many areas of the both ventricles.

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